

Acute aorto-iliac and mesenteric arterial thromboses as presenting features of COVID-19

SARS-CoV-2 infection has been reported to induce a hypercoagulable state,¹ which is associated with disease severity and poor outcomes.^{2,3} Rates of thromboembolic events and use of augmented thromboprophylaxis in patients with COVID-19 are therefore the subject of significant interest. However, the clinical manifestations of this procoagulant tendency are poorly defined. In this report, we describe two cases of major occlusive arterial events in previously asymptomatic individuals with confirmed COVID-19.

The first, a 60-year-old male, presented to hospital with sudden loss of sensation and power in both legs. Prior to this

he reported a dry cough, fever and general malaise for 2 weeks prior to hospital presentation, but no pre-existing symptoms of peripheral vascular disease. Examination revealed signs of bilateral acute lower limb ischemia with tachypnoea and tachycardia. Electrocardiogram was normal.

Initial laboratory studies revealed a mild leukocytosis (white cell count $13.1 \times 10^9/l$) with a mild lymphopenia ($1.1 \times 10^9/l$) and hyperfibrinogenemia (5.5 g/l). D-dimers were markedly elevated (23.6 mg/l, reference range 0–0.5 mg/l) but prothrombin time, activated partial thromboplastin time and platelet count were within normal limits.

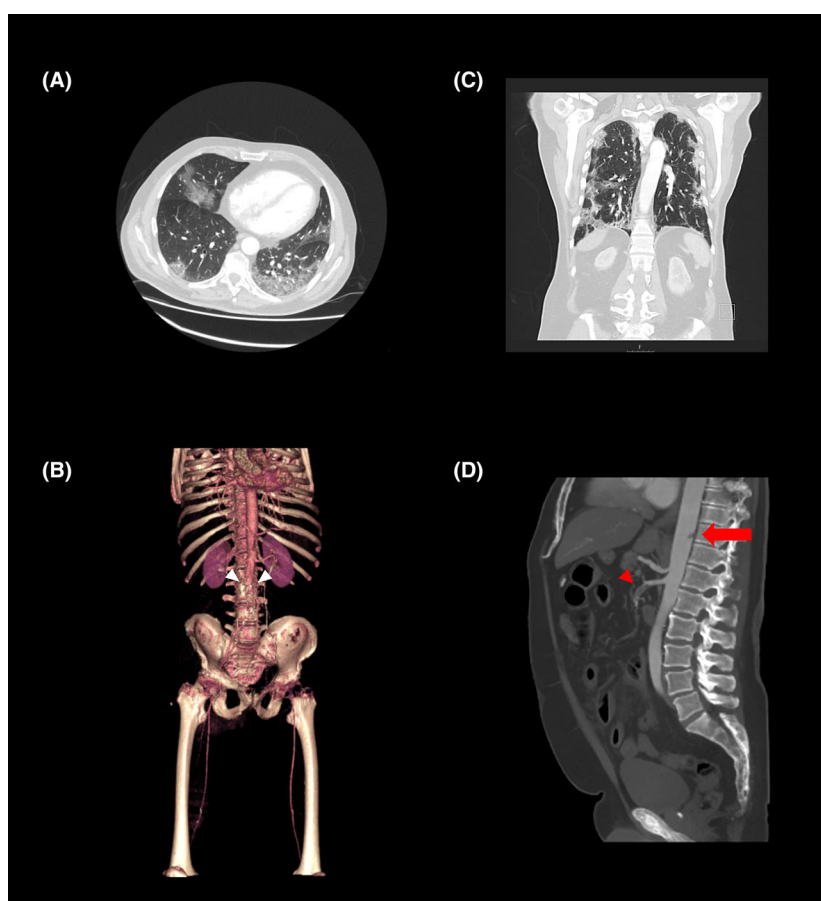


Fig 1. Arterial thrombotic events in patients with Covid-19. A and B: Computerised tomography (CT) of the chest (A) and angiography (B) in a 60-year-old man with cough, fever and leg paralysis. An abrupt occlusion of the infrarenal aorta is evident on angiography (white arrowhead, B). C and D: CT findings in a 75-year-old man with breathlessness and abdominal pain. Intraluminal thrombus in the descending thoracic aorta (red arrow) and superior mesenteric artery (red arrowhead) are indicated.

Chest radiology demonstrated diffuse bilateral consolidation and peripheral ground-glass opacification consistent with SARS-CoV-2 infection (Fig 1A). CT angiography revealed an acute thrombotic occlusion of the infrarenal aorta extending into the common iliac arteries (Fig 1B). Intraluminal calcific burden in the aorto-iliac segments was minimal with no stenotic disease. The patient underwent thromboembolectomy with retrieval of a large burden of acute thrombus, resulting in significant clinical and radiological improvement.

The second patient, a 75-year-old male with no comorbidities, attended the emergency department with abdominal pain and vomiting for 2 days, along with worsening of the cough and dyspnoea which he had experienced for the preceding 2 weeks. On arrival, his oxygen saturations were 88% on room air but the remainder of his observations were within normal limits. Electrocardiogram showed sinus rhythm with no ischemic changes. Laboratory studies demonstrated leukocytosis ($18.1 \times 10^9/l$), lymphopenia ($0.9 \times 10^9/l$) and a mild thrombocytosis ($497 \times 10^9/l$). D-dimer levels were elevated (3.2 mg/l) but troponin T, renal function and liver function were normal. Chest imaging was indicative of SARS-CoV-2 infection (Fig 1C). On CT angiography, intraluminal thrombus was present in the descending thoracic aorta with embolic occlusion of the superior mesenteric artery (Fig 1D) but no evidence of atherosclerosis. Catheter-directed thrombolysis was commenced but the patient developed worsening abdominal symptoms and underwent laparotomy, requiring resection of 150 cm of ischemic small bowel.

These cases illustrate that the prothrombotic sequela of Covid-19 are not confined to the venous circulation, and

macrovascular thrombi in the arterial circulation can occur in susceptible individuals during SARS-CoV-2 infection even in the absence of overt features of disseminated intravascular coagulation or severe respiratory manifestations.

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